Correspondence

Pulmonary atresia with ventricular septal defect and coronary artery fistula: a late presentation

Sir,

The case report by Vigneswaran and Pollock (1988;59:387-8) illustrates the continuing problems with the choice of the most appropriate words to describe congenital heart disease. They reported a patient with a solitary arterial trunk and complete absence of intrapericardial pulmonary arteries, with a connection between a coronary artery and the pulmonary arteries at the lung hilum. They described the arterial trunk as "truncus type IV". The problem with this description is that, in the absence of any intrapericardial pulmonary arteries, it is not possible to know whether initially, during development, the trunk had been an aorta or a common arterial trunk. For this reason, as suggested by Thiene and myself, it might be better simply to describe the great artery as a solitary trunk.

Also, Vigneswaran and Pollock describe the communication between the pulmonary arteries and the left coronary artery as a fistula. In that the structure functions as a conduit from the heart to the lung, it certainly is a fistulous communication. Previously, however, the term "coronary pulmonary arterial fistula" was used in cases such as those described by

Rastelli (reference 3 in Vigneswaran and Pollock's case report) and subsequently described in greater detail by Krongrad *et al.*² It seems to me that the communication in the case of Vigneswaran and Pollock is more akin to a systemic-pulmonary collateral artery arising from the left coronary artery and might better be described as such.

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References

- 1 Thiene G, Anderson RH. Pulmonary atresia with ventricular septal defect: anatomy. In: Anderson RH, Macartney FJ, Shinebourne EA, Tynan M, eds. Paediatric cardiology, volume 5. Edinburgh: Churchill Livingstone, 1983:80-101.
- 2 Krongrad E, Ritter DG, Kincaid OW. Aorticopulmonary tunnel: angiographic recognition of pulmonary atresia and coronary artery-to-pulmonary artery fistula. AJR 1973;119:498-502.

Spontaneous resolution of a dissection of the descending aorta after medical treatment with a β blocker and a calcium antagonist

Sir

We read with interest the report by Hoshino et al (1987;58:82-4) of spontaneous resolution of a dissection of the descending aorta. The computed tomographic scans in this case illustrate some of the poorly understood aspects of the pathogenesis of aortic dissection, which should be pointed out. In fig 1, the non-opacified crescentic area alongside the narrowed aortic lumen is characteristic of intramural haemorrhage without intimal tear. The false lumen did not become opacified because there was no intimal tear. Rupture of the vasa vasorum was the likely source of haemorrhage. As the haematoma was absorbed, the aortic lumen enlarged and the crescent

disappeared. This is seen in fig 2. These observations are confirmed by a recent report of 13 cases of aortic dissection without intimal breaks, diagnosed by computed tomography and magnetic resonance imaging. The diagnosis in such cases is often missed by angiography, as shown in another study of type III aortic dissections.

Intramural haemorrhage caused by rupture of the vasa vasorum is probably the initiating event in most aortic dissections. The intimal rupture is then a secondary event responsible for propagation of dissection and further complications. In the absence of rupture, complete healing is possible as shown by Hoshimo *et al.* This is particularly true for dissec-